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Case report

Native aortic valve thrombus as a source of embolisation into the coronary artery

Kamil Novobilský^{a,*}, Vladimír Kaučák^a, Jiří Bárta^b, Radim Kryza^a,
Jana Dvořáčková^c^aDepartment of Cardiology, Municipal Hospital Ostrava, Czech Republic^bDepartment of Cardiac Surgery, University Hospital Ostrava, Czech Republic^cDepartment of Pathology, University Hospital Ostrava, Czech Republic

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ABSTRACT

Authors present the case of 66-year-old patient after repeated systemic embolisations (lower extremity, axillary artery), admitted for inferior myocardial infarction. Coronary angiography demonstrated peripheral subtotal occlusion of posterior descending artery (PDA) of embolic origin. Transoesophageal echocardiography (TOE) revealed mobile mass on aortic valve, which was subsequently extirpated surgically. Histological examination described thrombus. Case report depicts the native aortic valve thrombus as a rare source of systemic or coronary embolisation. It simultaneously supports the indication of TOE at systemic embolisations of unknown source, even if transthoracic echocardiography (TTE) finding is normal, and shows its key role in diagnostic algorithm in similar events.

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Introduction

Embolisation as a mechanism of myocardial infarction is well known [1,2]. Prizel et al. found autopsy evidence of infarction caused by emboli in 5% hearts, selected for post mortem coronary angiogram because of known or suspected history of cardiovascular or pulmonary disease (that was 13% of all autopsy-confirmed infarctions) [1]. Its sources can be endocarditis (infective, nonbacterial thrombotic), thrombi from left-sided heart cavities, valves (native, prosthetic), paradoxical embolism, tumors (myxoma, fibroelastoma), or heart catheterization or surgery [3]. Nevertheless the native aortic valve

thrombosis as a source of embolisation into the coronary artery is uncommon.

Case report

Sixty six-year-old male with repeated systemic embolisation in his medical history, treated in other hospital: 2 years before current admission the acute occlusion of left low leg arteries, treated with partially successful local thrombolysis, and embolic occlusion of right axillary artery before 13 months, resolved by embolectomy. Subsequent hematologic examination for possible hypercoagulable state revealed just mild

* Correspondence to: Kardiologie, Městská nemocnice Ostrava, Nemocniční 20, 728 80 Ostrava, Czech Republic. Tel.: +420 596 194 279; fax: +420 596 192 343.

E-mail address: k.novobilsky@mnof.cz (K. Novobilský).

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hyperhomocysteinemia. Patient was temporarily treated with warfarin. There were still hypertension, celiac disease and mild chronic sideropenic anemia, related to celiakia, in his medical history. No thromboembolic event occurred in family history. Chronic medication included hydrochlorothiazide and amiloride in fixed-dose combination, oral iron supplement, allopurinol, betahistine. Warfarin was withdrawn 5 months before admission.

Patient was presented to our center from the emergency department of community hospital with repeated chest pain, the latest 2 days before admission, and new Q waves in leads II, III, aVF in his electrocardiogram. On admission the patient's state was stable, he was without dyspnea, his blood pressure was 120/80, heart rate 84 min⁻¹ and his lungs were clear within the auscultation, abdomen and lower extremities without abnormalities. The patient was referred for cardiac catheterization, coronary angiography demonstrated peripheral subtotal obstruction of PDA of embolic origin, only slight signs of atherosclerosis (Figs. 1 and 2). With regard to peripheral localization of thromboembolus, ECG signs of definite Q-infarction, clinical stabilization and absence of symptoms, the approach was conservative.

Laboratory tests showed microcytic, sideropenic anemia (hemoglobin 109...94 g/l), troponin I 25.401 ng/ml, iron level 3.4 µmol/l, renal insufficiency (kreatinin 162...149 µmol/l), total cholesterol 5.15 mmol/l, triacylglycerides 1.78 mmol/l, LDL-cholesterol 3.22 mmol/l, HDL-cholesterol 0.91 mmol/l. Transthoracic echocardiography reveals left ventricle ejection fraction 45%, inferior wall hypokinesis, mild aortic and tricuspidal insufficiency (trace regurgitation), mitral regurgitation I/IV. Chest X-ray was normal. Within search for a source of embolisation TOE was performed and showed echogenic mobile ovale mass (Fig. 3) attached by a stalk to the right coronary leaflet of aortic valve, 27×10 mm, with trace regurgitation. There was mitral regurgitation II/IV, left atrium including the appendage without clots. Our differential diagnosis of the mass included fibroelastoma, other type of tumor or thrombus. Vegetation was considered unlikely.

Additional examinations performed within the hospitalization: abdominal ultrasonography revealed a "bright" liver



Fig. 1 – Right coronary artery with peripheral subtotal obstruction in PDA (arrows).

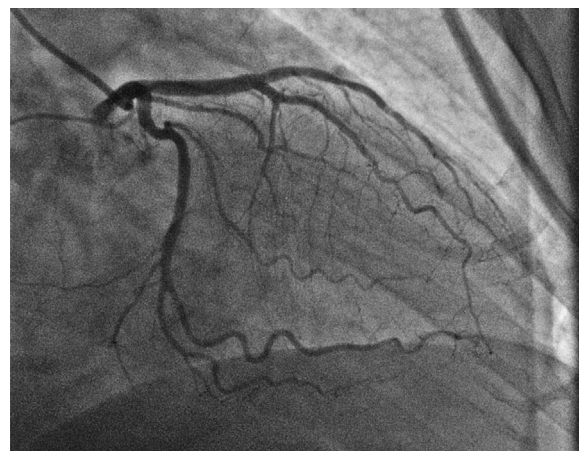


Fig. 2 – Left coronary artery.

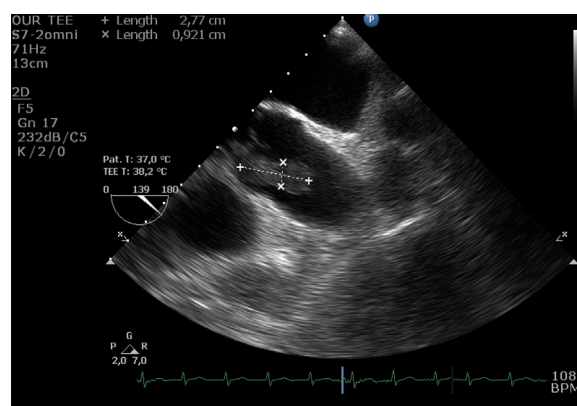


Fig. 3 – Transoesophageal finding of mass attached to the aortic valve.

with increased echogenicity, chronic nephropathy on the left, fibrogastroscopy described sliding hiatus hernia, suspected gastroesophageal reflux and atrophic mucosa in duodenum ("mosaic" pattern), suspected of celiakia.

The course of hospitalization, lasting 5 days, was uncomplicated, no further systemic embolisation occurred. Anticoagulation with low-molecular weight heparin was initiated, followed by warfarin in usual way.

4 weeks later patient underwent surgery: in cardiopulmonary bypass the mass of aortic valve was extirpated, macroscopically described as fragile, crumbly, jelly-like. Based on perioperative TOE, mitral valve anuloplasty was performed simultaneously. Histological finding depicts mixed, organized, mainly red thrombus (Fig. 4). Postoperative course was uneventful, patient was discharged on 10th day with warfarin, intended for lifelong. Currently (9 months after surgery) patient is doing well, managed by cardiologist and angiologist in outpatient clinics.

Discussion

The native aortic valve thrombus as a cause of peripheral embolisation, or more precisely myocardial infarction, is rare

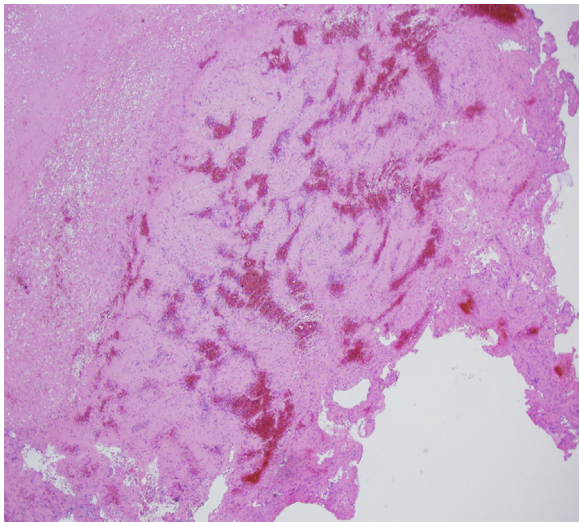


Fig. 4 – Histological finding: particle formed of structures of mixed, predominantly red thrombus pervaded with dispersed, in some places merging granulocytotic cellulisation. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

[4–6], in most described cases associated with hyperthrombogenic state (antiphospholipid syndrome, protein S deficiency) [7] or with systemic connective tissue disease (systemic lupus erythematosus), alternatively formed on diseased valve (calcifications, amyloidosis) [8,9] or as a consequence of trauma (surgery, catheterization) [10].

In our case the exact mechanism responsible for thrombus formation was not clearly identified. Hematological examination revealed just mild hypercoagulable state (mild hyperhomocysteinemia, heterozygous for the methylentetrahydrofolate reductase gene mutation), only trace regurgitation on the aortic valve was discovered as an organic change. Celiac disease is known as a potential risk factor for venous thrombosis [11,12], however only a few cases reported patients with thrombosis in arterial bed, and none of them refers the intracardial or valvular thrombus as a source of embolisation [13,14]. There could be a theoretical link of celiac disease as an autoimmune disorder to the non-bacterial thrombotic endocarditis, but that was not proved in the presented case.

Search for a potential cardiac source of systemic embolism represents one of principal indications for transoesophageal echocardiography, that plays then key role in diagnostic algorithm of embolic events, even with normal finding in transthoracic examination [15–17]. The same was true in our case of patient after repeated systemic embolisation (arteries of lower extremity, axillary artery, right coronary artery), where transoesophageal echocardiography revealed the mass on aortic valve, undiscovered in transthoracic way. The histological finding after surgical resection confirmed characteristics of a thrombus.

Conclusion

Spontaneous native aortic valve thrombosis is infrequent. It should be taken into the consideration in the differential diagnosis of valve-associated masses and causes of systemic embolism. Transoesophageal echocardiography is of crucial importance in its detection.

Conflict of interest

No conflict of interest.

Funding body

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Ethical statement

The research was done according to ethical standards.

Informed consent

The patient described in this case report has been informed and gave his/her consent for publication.

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